and would be able to improve the diagnosis and monitor the progression of the disease.

IC-P-011 A PERIPHERAL DIAGNOSTIC FOR ALZHEIMER'S DISEASE WITH 100% SPECIFICITY AND 94% SENSITIVITY IN PRELIMINARY TESTING

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Background: Presently, Alzheimer's disease (AD) can only be diagnosed with certainty at autopsy. Although imaging methods are beginning to approach this standard, they are expensive and, for some methods, invasive. We have therefore attempted to develop a simple, safe, inexpensive, and highly accurate peripheral blood diagnostic for AD. We have previously observed a significant hypomethylation of DNA in AD cortex. Because this change was so dramatic, and because it extended to both neurons and glia, we speculated that similar alterations might also occur in peripheral leukocytes. **Methods:** Venous blood samples were taken from 17 living AD and 19 living ND subjects. Diagnoses were based on standard NIA AD Center criteria. Under thorougly blinded conditions, leukocytes were isolated from the buffy coat, immunoreacted with antibodies to 5-methylcytosine, 5-methylcytidine, DOC1, or HDAC1, and evaluated qualitatively as ''substantial staining'' or ''sparse staining''. **Results:** Based on obvious staining patterns (e.g., sparse immunoreactivity for DOC1 in AD cases and substantial immunoreactivity in ND cases), AD was correctly diagnosed in 16/17 cases (94% sensitivity) and ND was correctly diagnosed in 19/19 cases (100% specificity). **Conclusions:** Additional subjects are needed to confirm these results. Perhaps more important, they also need to be extended to MCI and non-AD dementias using quantitative rather than qualitative methods. Such research is now ongoing.

IC-P-012 A CASE REPORT OF ALZHEIMER'S DISEASE WITH PRESENILIN-1 MUTATION (MET233LEU) SHOWING A NEW PHENOTYPE

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Background: Two cases of the phenotypes (clinical symptoms and neuroimaging abnormalities) of early onset Alzheimer disease (AD) with presenilin-1 (PREN1) mutation (Met233Leu) have been reported. Both of the patients showed the core features of frontotemporal dementia (FTD) as well as those of AD. In both cases, images of MR and SPECT were not analyzed in detail. We have experienced a 38 years old female patient with familial AD associated with PREN1 mutation (Met233Leu). **Methods:** We comprehensively performed neuropsychological tests, neurological examinations and genetic examinations. In addition, we performed brain MRI and cerebrum blood perfusion SPECT, and statistically analyzed MRI data using VSRAD and SPECT data using 3D-SSP. **Results:** She showed memory impairment when she was 36 years old and the symptom gradually worsened. She came to our hospital two years later. At that time, she showed severe impairments of memory and visual-spatial perception. However, she had none of the core features of FTD; social unawareness or impropriety, abnormal personal regulation or apathy, emotional blunting, loss of empathy, and decreased insight. Her neurological tests revealed no abnormalities, including myoclonus, seizures, or spasticity. Her MMSE score was 12/30 at her first examination. At 16 months later, her MMSE score was 5/30. But even then she remembered her doctor's name and face after one month interval, was aware of her memory impairment, and showed no features of FTD, nor myoclonus, seizure. In EEG, her basic rhythm is 6-7 Hz with frequent contaminations of 3-4 Hz slow waves. Her brain MR images showed no remarkable atrophy in all regions including hippocampus and parietal lobe. The VSRAD showed no atrophy in the parahippocampal gyrus. The 3D-SSP showed remarkable regional hypoperfusion in the lateral frontal, parietal, precuneus, and posterior cingulated gyrus, but no hypoperfusion in the hippocampal, parahippocampal, and orbital gyri. A phenotype of this patient shows no features of FTD. APOE was 3/3, HLA-A was A31/A24, and KIBRA(rs17070145) was T/T. **Conclusions:** Our AD patient with PREN 1 mutation (Met233Leu) did not have any symptoms observed in FTD patients, unlike the other patients with the same mutation reported before.

IC-P-013 THE AIBL STUDY: BASELINE DATA FROM A MULTICENTER, PROSPECTIVE LONGITUDINAL STUDY OF AGEING IN 1,100 VOLUNTEERS

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Background: The ability to diagnose Alzheimer's disease (AD) in its earliest stages will greatly optimize treatment and preventative strategies and may potentially delay or prevent disease progression. The Australian Imaging Biomarkers and Lifestyle Flagship Study of Ageing (AIBL) is a three-year prospective longitudinal study of over 1100 volunteers from a cross-section of Australia's elderly population. This study aimed to improve understanding of the pathogenesis, early clinical manifestation, and diagnosis of AD and identify intervention targets which may delay or eventually prevent disease onset. **Methods:** The cohort comprises 1112 volunteers aged over 60 years [211 patients with AD (mean age 78.0 + 8.6 years), 133 patients with MCI (mean age 75.7 + 7.6 years), and 768 healthy volunteers (HV; 70.0 + 7.0 years)]. All volunteers completed lifestyle questionnaires and underwent comprehensive clinical and neuropsychology assessment. An 80 ml blood sample was provided for clinical pathology, biomarker analysis, and storage in liquid nitrogen. 286 participants received a [C-11] PIB-PET scan (a measure of in vivo amyloid) and a MRI scan. In addition, 100 received scans of body composition (DEXA) and 91 participated in actigraph monitoring of activity levels. **Results:** AD patients performed worse on all neuropsychological measures compared to both HV and MCI groups, and MCI patients showed greater impairment than HVs (all $p < 0.05$). Neuroimaging subgroup results revealed a significant difference between groups in the PiB +ve volunteers (98% of AD patients, 64% of MCI patients and 29% of HVs). HVs with an apolipoprotein-E (ApoE) $\epsilon 4$ allele were significantly more likely to be PiB+ve than ApoE $\epsilon 4$ negative HVs (49% compared to 21%, respectively). **Conclusions:** Cross sectional analysis of baseline data will reveal links between cognition, brain amyloid burden, structural brain changes, biomarkers, and lifestyle. An 18-month follow-up will reveal risk factors associated with cognitive decline and identify early diagnostic indicators of AD. These findings will assist development of techniques to identify factors which may delay onset of AD, and provide a cohort suitable for early intervention studies.

IC-P-014 THE FUNCTIONAL IMPACT OF CEREBRAL WHITE MATTER HYPERINTENSITIES IN THE ELDERLY

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